INFLUENCE OF BRAIN MONOAMINES ON THE COURTSHIP BEHAVIOR OF MALE RING DOVES (STREPTOPELIA RISORIA)

by

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Date: Feb. 16, 1978

Approved:

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Dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Psychology in the Graduate School of Duke University

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ABSTRACT

(Psychology-Physiological)

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INFLUENCE OF BRAIN MONOAMINES ON THE COURTSHIP BEHAVIOR OF MALE RING DOVES (*STREPTOPELIA RISORIA*)

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Male ring doves received intracerebral implants of various pharmacological agents to determine whether brain monoamines are involved in the regulation of courtship behavior and to elucidate the physiological nature of any monoaminergic effects. L-norepinephrine, d-norepinephrine, phenylephrine, isoproterenol, tyramine, serotonin, parachlorophenylalanine, barium chloride, and papaverine effectively suppressed the bow-cooing and nest-soliciting courtship displays when placed within a circumscribed ventromedial forebrain region having the preoptic-anterior hypothalamic area as its approximate center. The courtship displays were little or not suppressed by desipramine, dopamine, alpha-methyl-para-tyrosine, phentolamine, LB 46 (a beta-adrenergic receptor antagonist), gamma-amino-butyric acid, and glutamic acid. Although non-specific drug effects may have accounted for a large degree of the

observed behavioral suppression, the differential effectiveness of the drugs on bow-cooing and nest-soliciting suggests that nest-soliciting may be specifically inhibited by an alpha-adrenergic neural system located within the preoptic-anterior hypothalamic area of the brain.

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R. C.

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INTRODUCTION

It has been suggested that brain monoamines influence sexual behavior in male mammals (Sandler & Gessa, 1975). A serotonergic involvement was originally implicated by observations of marked mounting behavior among male rats (Sheard, 1969; Shillito, 1970; Tagliamonte, Tagliamonte, Gessa, & Brodie, 1969), rabbits (Gessa, 1970; Perez-Cruet, Tagliamonte, Tagliamonte, & Gessa, 1971), and cats (Ferguson, Henriksen, Cohen, Mitchell, Barchas, & Dement, 1970; Hoyland, Shillito, & Vogt, 1970) following treatment with parachlorophenylalanine (PCPA), a drug which inhibits the synthesis of serotonin at the tryptophan hydroxylase step. 5-Hydroxytryptophan, the immediate precursor of serotonin, reversed this behavioral effect. Other treatments which reduce serotonergic function, e.g., limitation of dietary intake of tryptophan, the amino acid precursor of serotonin (Gessa & Tagliamonte, 1975), and intraventricular administration of 5, 6-dihydroxytryptamine, a drug which destroys serotonergic neurons (DaPrada, Carruba, O'Brien, Saner, & Pletscher, 1972), also produced male-to-male mounting in rats under conditions in which this behavior does not normally occur.

PCPA was also found to increase the incidence of heterosexual

behavior in sexually sluggish, inexperienced male rats (Tagliamonte, Tagliamonte, & Gessa, 1971) and in castrated rats receiving suboptimal doses of testosterone (Malmnäs, 1973; Malmnäs & Meyerson, 1971). Moreover, PCPA enhanced the sexual performance of sluggish and normal rats, e.g., decreased intromission frequency, ejaculation latency, and post-ejaculatory interval, and increased the number of ejaculations (Gessa & Tagliamonte, 1974b; Mitler, Morden, Levine, & Dement, 1972; Salis & Dewsbury, 1971; Soulairac & Soulairac, 1975). However, the performance of sexually vigorous animals was not enhanced by PCPA (Whalen & Luttge, 1970). In contrast to PCPA, 5-hydroxytryptophan treatment abolished spontaneous sexual behavior with a receptive female rat (Malmnäs, 1974; Tagliamonte, Fratta, Mercuro, Biggio, Camba, & Gessa, 1972). However, 5-hydroxytryptophan has also been shown to facilitate sexual performance in the rat by decreasing the ejaculation latency, the number of intromissions to obtain an ejaculation, and the post-ejaculatory interval (Soulairac & Soulairac, 1975).

In other species tested, PCPA did not further facilitate heterosexual performance in vigorous male cats (Zitrin, Beach, Barchas, & Dement, 1970) and produced only inconsistent results in rhesus monkeys Perachio & Marr, cited in Zitrin, Dement, & Barchas, 1973; Redmond, Maas, Kling, Graham, & Dekirmenjian, 1971). The sexual effect of PCPA in human males was examined in migraine sufferers who complained of strongly reduced or abolished libido (Sicuteri, 1974; Sicuteri,

Del Bene, & Anselmi, 1975). Neither PCPA nor testosterone alone had a significant effect on the number of daily erections. However, the combination of PCPA and testosterone produced a significant increase in the number of erections.

Evidence has also been presented for a dopaminergic role in the regulation of sexual behavior. Either L-dihydroxyphenylalanine (L-DOPA), the immediate precursor of dopamine, or apomorphine, a specific stimulator of central dopaminergic receptors, enhanced the incidence of male-to-male mounting in rats (DaPrada, Carruba, Saner, O'Brien, & Pletscher, 1973) and of heterosexual copulatory behavior in male rats with a low baseline of sexual performance (Gessa & Tagliamonte, 1974a, 1974b, 1975; Malmnäs, 1973, 1974). Drugs which decrease dopaminergic activity, e.g., the catecholamine synthesis inhibitor alpha-methyl-para-tyrosine, and the dopamine receptor blockers haloperidol and pimozide, have been found to decrease the incidence of heterosexual behavior in vigorous and sluggish male rats (Gessa & Tagliamonte, 1975; Malmnäs, 1973, 1974). The data concerning the effect of dopamine on the individual components of copulatory behavior in rats have been inconsistent: L-DOPA treatment has either facilitated (Gessa & Tagliamonte, 1974a), weakly inhibited (Gray, Davis, & Dewsbury, 1974), or not affected (Hyyppä, Lehtinen, & Rinne, 1971) performance.

In human Parkinsonian patients being treated with L-DOPA to

alleviate their motor symptoms, sexual excitement and enhanced libido have been among the observed clinical side effects of the drug (Bowers, Van Woert, & Davis, 1971; Hyyppä, Rinne, & Sonninen, 1970; Mars, Libman, Schwartz, Gillo-Joffroy, & Barbeau, 1972). L-DOPA treatment in human males suffering organic impotence slightly increased penile erections and the frequency of spontaneous erections but did not lead to satisfactory sexual intercourse (Benkert, 1972).

Although less attention has been devoted to the possible influence of norepinephrine on sexual behavior, Soulairac and Soulairac (1975) found that systemic administration of norepinephrine in rats abolished sexual behavior, and that dibenamine, an alpha-adrenergic receptor blocker, increased the number of ejaculations and reduced the ejaculation latency and number of intromissions to achieve an ejaculation. Propranolol, a beta-adrenergic receptor blocker, had no effect. However, Malmnäs (1973, 1974) did not find an increase in the incidence of heterosexual mounting following administration of either phenoxybenzamine, another alpha-adrenergic blocker, or propranolol in castrated rats receiving suboptimal doses of testosterone.

In the course of a preliminary attempt in this laboratory to determine whether monoamines influence reproductive behavior in an avian species, it was found that norepinephrine implants made in the vicinity of the preoptic region of the brain suppressed the nest-soliciting courtship display in the male ring dove.

The male ring dove normally performs two prominent courtship displays in initial interactions with a female, bow-cooing and nest-soliciting. Bow-cooing is characterized by repetitive bowing to the female, with each bow accompanied by a vocalized "coo." Nest-soliciting is distinguished by the male's oblique posture, head lowered and tail raised, as it vibrates its wing tips and emits a "coo" every few seconds (see Miller & Miller, 1958, for complete description).

Both bow-cooing and nest-soliciting are dependent upon androgen since castration abolishes them and testosterone replacement reinstates them (Erickson & Lehrman, 1964; Hutchison, 1970a). Moreover, both displays seem to share a common anatomical basis; discrete implants of testosterone made in the preoptic or anterior hypothalamic regions of the brain in castrated animals restore both displays (Barfield, 1971; Hutchison, 1967, 1970b, 1971).

However, the performance levels of these displays diverge during the course of the reproductive cycle and in different social and hormonal conditions. During the first few days of pairing with a female both displays decline with time, but bow-cooing declines more rapidly than nest-soliciting (Lovari & Hutchison, 1975; Martinez-Vargas & Erickson, 1973). A male bow-coos more to a strange female than to a familiar mate (Erickson, 1973; Erickson & Morris, 1972), and nest-solicits less to a female that has been previously associated with another male than to one that has not (Erickson & Zenone, 1976). Furthermore, progesterone

treatment suppresses bow-cooing but does not affect nest-soliciting (Erickson, Bruder, Komisaruk, & Lehrman, 1967), and estrogen administration in a castrated male reinstates nest-soliciting but not bow-cooing (Hutchison, 1970a). Bow-cooing and nest-soliciting also appear to have different sensitivity thresholds to androgen (Hutchison, 1974a, 1974b). Thus, after castration bow-cooing declines more rapidly than nest-soliciting. If testosterone is administered shortly after castration, both displays are reinstated. However, when testosterone is given one month after castration, only nest-soliciting is reinstated. In addition, small implants of testosterone within the preoptic-anterior hypothalamic area reinstate only nest-soliciting in castrated animals, but larger implants are needed to restore both displays (Hutchison, 1970b).

Thus, while bow-cooing and nest-soliciting may share a basic dependence upon androgen uptake in preoptic-anterior hypothalamic sites, the fact that these displays can vary independently and the possibility that norepinephrine selectively affects nest-soliciting suggest that bow-cooing and nest-soliciting may be under different neurochemical control. Hence, the present experiment was an attempt to confirm the norepinephrine effect on nest-soliciting, to examine the physiological nature of this effect by observing the behavioral effects of other adrenergic drugs and control substances for norepinephrine, and to determine whether serotonin and dopamine also influence courtship displays in the ring dove.

METHOD

Animals and Maintenance

The subjects were 174 male ring doves, 110-180g in weight, which had completed one or more successful breeding cycles. Prior to being used in the experiment, they had been visually isolated from other birds for at least two weeks.

Female test partners were selected only if they displayed behavior patterns characteristic of the early part of the normal breeding cycle, i.e., the low responsiveness and approaching behavior of stages I, II, and III in the Cheng (1973) classification.

All birds had been reared in the colony and throughout the experiment were housed individually in wooden cages (41 x 45 x 36 cm^3) with wire mesh fronts. Although unable to see other birds, they could hear colony sounds. They were exposed to a 14L:10D diurnal lighting cycle. Food, water, and grit were provided ad libitum except for 24 hours prior to surgery.

Cannulae and Drugs

Drugs were applied to discrete brain regions by means of a chronically implanted cannula system (Plastic Products, Roanoke, Va.). The

system consisted of a guide cannula (22 gauge) with a threaded sleeve at one end, a removable internal cannula (28 gauge), and a cap that screwed onto the sleeve of the guide cannula. The internal cannula obtained from the manufacturer was shortened, making its tip flush with that of the guide cannula when the parts were assembled.

The drugs in powdered form were delivered to the brain by tapping them into the lumen of the internal cannula, inserting this cannula into the implanted guide cannula, and allowing the drug to diffuse to the surrounding brain tissue. The lumen was filled by dipping the cannula tip five times into a layer of the drug formed by pressing a small amount of the crystalline drug between the two halves of a folded glassine sheet of paper.

The drugs tested and the time interval between drug application and behavioral testing were the following: l-norepinephrine bitartrate (Adams)--10 min; d-norepinephrine bitartrate (Adams)--10 min; phenylephrine hydrochloride (Gane's)--10 min; isoproterenol hydrochloride (Gane's)--10 min; tyramine hydrochloride (Sigma)--15 min; desipramine hydrochloride (USV)--1.5 h; dopamine hydrochloride (Sigma)--10 min; dl-α-methyl-p-tyrosine methyl ester hydrochloride (Sigma)--4 h and 1.5 h; phentolamine hydrochloride (CIBA)--15 min; LB 46 (dl-4-[2-hydroxy-3-isopropylaminopropoxy]-indole; Sandoz)--15 min; serotonin creatinine sulfate (Pfaltz & Bauer)--10 min; dl-p-chlorophenylalanine methyl ester hydrochloride (Calbiochem)--every 24 h for 3 days and

fourth application 4 h before test; barium chloride (Sigma)--10 min; papaverine hydrochloride (Lilly)--10 min; γ -amino-n-butyric acid (Sigma)--10 min; L-glutamic acid (Sigma)--10 min.

At the end of a test, the lumen of the internal cannula was cleaned and dried by immersing the tip into successive baths of water, 70% ethanol, and 100% ethanol.

Surgery

The doves were castrated and implanted with the cannula system in a one-stage operation while under L.A. Thesia or Equithesin anesthetic (0.25/100g body weight). Bilateral castration was performed first. Each bird was then prepared for implantation by plucking the head and ear feathers and applying Neosporin ointment in the ears. After placing the head in the stereotaxic apparatus, the skull was exposed and scraped lightly with a dental pick to facilitate adherence of the dental cement.

The stereotaxic coordinates were based on the Karten and Hodos atlas of the pigeon (1967). The pigeon coordinates were converted to ring dove coordinates by applying the following formulae which had been determined in a preliminary histological investigation: anterior-posterior plane, ring dove (RD) = 0.67 pigeon (P); dorso-ventral plane, RD = 0.75 P; medio-lateral plane, RD = 0.90 P.

Once the head was properly positioned, a burr hole was made in the skull, the dura cut, and the guide cannula implanted. Since preliminary

tests suggested that norepinephrine implants made in the vicinity of the preoptic area could change courtship performance, a large proportion of cannulae were aimed for this region. In addition, cannulae were also implanted in many other brain sites. Unilateral implants were made in either the left or right side of the brain, half the birds receiving implants in the left side and half in the right side. Once the cannula was in place, dental cement (Fastcure) was molded conically around the upper end of the cannula to secure it to the skull. The inner cannula was inserted into the guide cannula and capped at this time.

The birds were weighed every day throughout the experiment beginning on the day of pretest. After surgery they returned to their normal weights within one to three weeks.

Behavioral Testing

All behavioral tests were conducted in a cage located in the same colony room in which the birds were housed. The test cage, twice the size of the individual cages (82 x 45 x 36 cm^3), contained a nest bowl, pine needles, a perch, food, and water. Before the test, the cage was divided in half by an opaque partition, and the male and female were placed in the separate halves for an acclimation period of at least 10 min.

At the scheduled time before the test (determined by the drug to be tested; see section on cannulae and drugs), the male's inner cannula was removed and replaced by a drug-filled or blank cannula. All blank controls were applied 10 min before the test.

The male was tested with a different female on each test. The following behavioral patterns performed by the male were recorded for a period of 15 min: chasing, kah-calling, bow-cooing, wing-flipping, nest-cooing, preening, heteropreening, pecking, slapping, mounting, copulation, eating, and drinking. A full description of these behavioral patterns can be found in Miller and Miller (1958). However, only the results on courtship displays and preening are reported since these were the only behavioral patterns that occurred frequently enough under the present testing conditions to provide reliable data analysis. The behavioral data were recorded as follows: (1) chasing--the number of 1/2-min intervals in which the male chased the female or kah-called; (2) bow-cooing--(a) the number of times that the male bowed and cooed at the female and (b) the number of bouts of bow-cooing; (3) nest-soliciting--(a) the number of 1/2-min intervals in which the male wing-flipped and (b) the number of times he nest-cooed; (4) preening--the number of 1/2-min intervals in which the bird preened itself.

Experimental Procedure

Screening test. The courtship behavior of the male was observed on one occasion before surgery. Only birds which showed both bow-cooing and nest-soliciting displays were used in the experiment.

Surgical and hormonal preparation. One to four days after the pre-test the doves were castrated and implanted with the cerebral cannula

assembly. Daily treatment with 100 μg testosterone proprionate (dissolved in 0.10 cc sesame oil and injected intramuscularly) was begun 48 h after surgery and continued until the end of the experiment.

Drug tests. Experimental behavioral tests were begun three weeks following the initiation of testosterone treatment. These tests, given every four days, were made under alternating blank and drug conditions. Each bird was tested with an average of four drugs and almost all received l-norepinephrine. The order of drug administration among the birds was randomized.

Histology. At the end of the test series, the males were anesthetized and perfused with 10% formalin through the left ventricle of the heart. The brains were removed and fixed in 10% sugar formalin. Frozen sections were cut at 40 μ and stained with cresyl violet. A small number of sections were stained with oil red O and counterstained with hematoxylin (Cholewiak, Butcher, & Kettlewell, 1968).

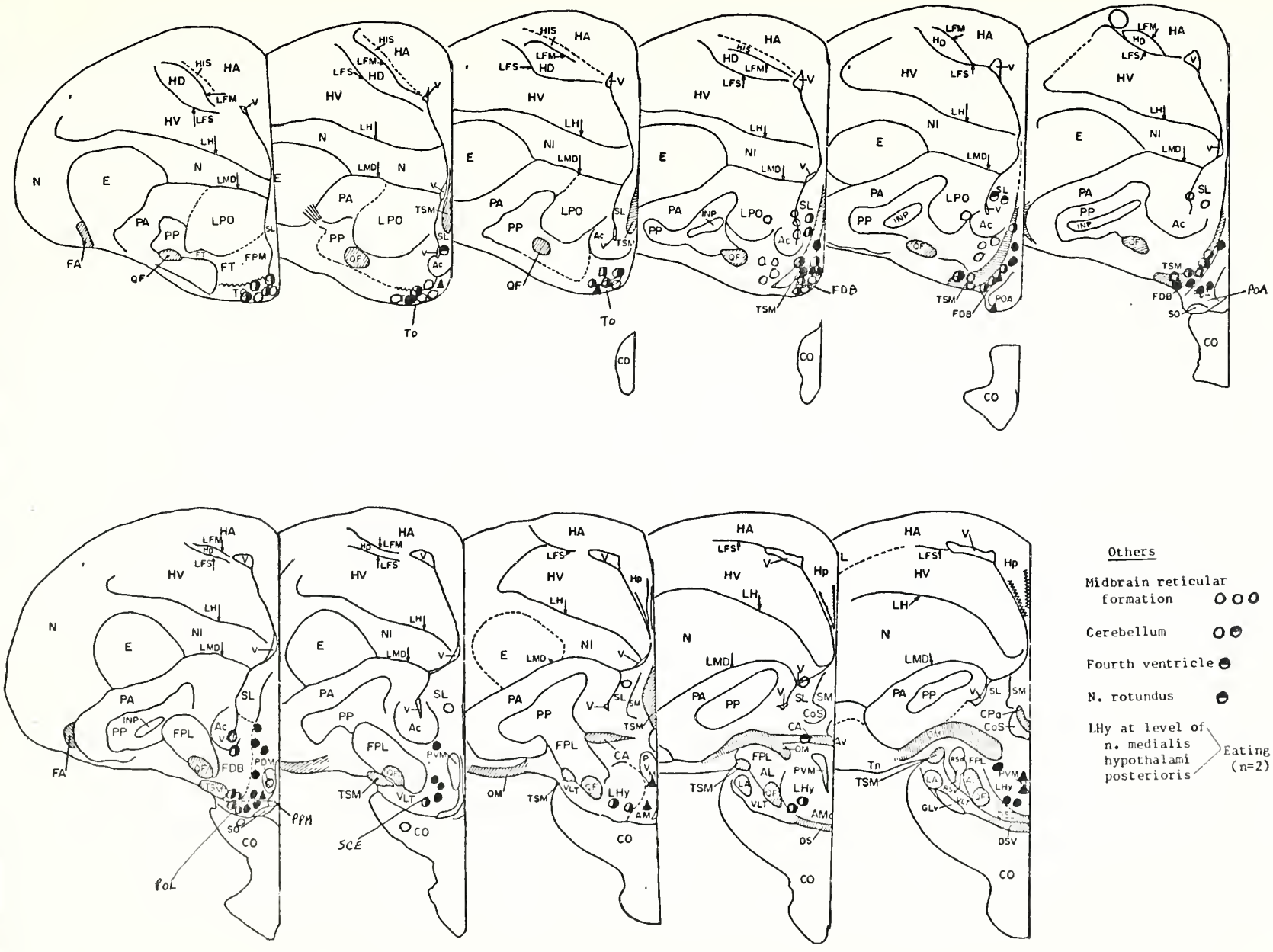
RESULTS

Of 174 experimental birds, data on 28 are omitted for the following reasons: cannulae penetrated base of brain (n = 17); implant dislodged (n = 7); inner cannula stuck in guide cannula (n = 3); eye infection (n = 1). The behavioral data of an additional 30 birds were excluded because these birds failed to meet a criterion of behavioral stability in control blank tests. The criterion, that both bow-cooing (BC) and nest-soliciting (NS) be displayed on more than 60% of the blank tests, was established to ensure that behavioral results obtained under drug conditions would be tested against a stable level of baseline responding.

Anatomical Results

Figure 1 illustrates the anatomical locations of cannula implants of 116 birds in which drug effects were analyzed. Almost all implants were found scattered in a circumscribed area of the forebrain with the preoptic region as its approximate center. The most peripheral implants in this area were located in the tuberculum olfactorium (TO) anteriorly, nucleus rotundus posteriorly, septum lateralis (SL) dorsally, chiasma opticum (CO) ventrally, and paleostriatum augmentatum (PA) laterally. The few implants located outside the forebrain were placed in the midbrain

Figure 1. Cannula implant locations in 116 experimental birds and locations of l-norepinephrine placements associated with various courtship responses. ● = BC and NS suppression; ◑ = NS suppression alone; ◓ = BC suppression alone; O = no change in behavior; ▲ = inactivity; ◨ = BC enhancement and NS suppression. Ac = n. accumbens; AM = n. anterior medialis hypothalami; CA = commissura anterior; CO = chiasma opticum; FDB = fasciculus diagonalis Brocae; LHy = n. lateralis hypothalami; LPO = lobus parolfactorius; N = neostriatum; PA = paleostriatum augmentatum; POA = n. preopticus anterior; POL = n. preopticus lateralis (Huber & Crosby, 1929); POM = n. preopticus medialis; PP = paleostriatum primitivum; PPM = n. preopticus paraventricularis magnocellularis; PVM = n. periventricularis magnocellularis; SCE = stratum cellulare externum (Huber & Crosby, 1929); SL = n. septalis lateralis; SO = n. supraopticus; TO = tuberculum olfactorium; TSM = tractus septomesencephalicus; V = ventricle; VLT = n. ventrolateralis thalami. (Reprinted from Karten, H. J., & Hodos, W. A stereotaxic atlas of the brain of the pigeon [Columba livia] by permission of publisher. Copyright 1967 by Johns Hopkins University Press, Baltimore 21218.)



Others

Midbrain reticular formation

Cerebellum

Fourth ventricle

N. rotundus

LHy at level of n. medialis hypothalami posterioris — Eating (n=2)

reticular formation (n = 3), fourth ventricle (n = 1), and cerebellum (n = 2).

Criterion for Drug Effect

Since no two birds had implants in precisely the same neuroanatomical site, there was no a priori basis on which to group them for analysis of drug effects. Although implants within the same cytoarchitectonic anatomical area might be expected to have similar effects, it is quite possible that these implants, differing only slightly in location, impinge on entirely different chemically coded systems. Conversely, widely separated implants could affect the same wide-ranging chemically coded system. Therefore, a criterion was chosen upon which to base a judgment that any observed change in a bird's behavioral response after drug application would not be due to normal variation in the level of performance. A difference of greater than 70% between drug condition and median blank performance was selected as the criterion. This criterion was applied to BC and NS separately. Since NS consists of wing-flipping and nest-cooing behavioral patterns, both component patterns were required to meet the criterion for NS to be considered as having changed.

The criterion was generated by comparing the individual performance of the 116 experimental birds on a single blank test with each bird's median performance on the remaining blank tests. As a control for order effects, performance on the first blank test was selected for

comparison in the first bird, second blank in the second bird, and so on until the fifth blank in the fifth bird. This numerical sequence was then repeated among the remaining birds. The mean difference between performance on the single blank test and the median performance on the remaining blank tests among the birds for the BC display was 36% (range 0-87%) and for NS was 27% (range 0-88%). Given the rather high upper limit of the range of differences, the 70% criterion was established since relatively few birds exceeded this degree of difference. The percentage of birds that did so provides an estimate of the expected error due to this criterion level. Performance of BC on the single blank test was enhanced in 2% and suppressed in 4% of the birds. The single blank performance of 94% of the birds failed to meet the criterion level of difference in BC. Performance of NS on the single blank test was enhanced in 1% and suppressed in 4% of the birds. The single blank performance of 95% of the birds failed to meet the criterion level of difference in NS.

l-Norepinephrine

Application of l-norepinephrine (l-NE) produced the following behavioral effects in 116 birds: suppression of both BC and NS (n = 26); suppression of NS alone (n = 33); suppression of BC alone (n = 7); enhancement of BC and suppression of NS (n = 1); inactivity (n = 9); eating (n = 2); and no change in behavior (n = 38).

The effects of l-NE on chasing and preening behavior are illustrated in Table 1. Separate comparisons were made between median

Table 1

Performance of Chasing and Preening in Blank and l-Norepinephrine Conditions by Birds Showing Various Bow-Cooing, Nest-Soliciting or Activity Responses to l-Norepinephrine

Group	Blank		l-Norepinephrine		
	Median	Range (in 1/2-min intervals)	Median	Range	p
Chasing					
BC & NS Suppr (n = 26)	4	1.5-13	2	0-9	< .00006
NS Suppr (n = 33)	6	2-14	5	1-19	> .10
BC Suppr (n = 7)	4	2-9	3	0-7	> .10
No change (n = 38)	3.5	2-12	4.3	2-14	> .10
Inactive (n = 9)	5.5	2-13	1.3	0-4	< .01
Preening					
BC & NS Suppr (n = 26)	7.3	0-16.5	13	1-27	< .00006
NS Suppr (n = 33)	7.5	0-16	12.5	0-20	< .00006
BC Suppr (n = 7)	7	0-15	8.5	0-19	> .10
No change (n = 38)	6.5	0-18	7	0-19	> .10
Inactive (n = 9)	7	3-13	12	3-17	> .10

blank and drug condition performance of birds showing the different major responses to 1-NE. Chasing was significantly less in the 1-NE than blank condition in birds that showed both BC and NS suppression (Wilcoxon matched-pairs signed-ranks test, $z = 4.70$, $p < .00006$). Chasing was not significantly changed in birds showing only NS suppression (Wilcoxon test, $z = 0.62$, $p > .10$), only BC suppression (Wilcoxon test, $T = 4$, $p > .10$), or no change (Wilcoxon test, $z = 1.03$, $p > .10$). Preening, in contrast to chasing, was enhanced in birds in which 1-NE suppressed both BC and NS (Wilcoxon test, $z = 4.58$, $p < .00006$) and also in birds in which 1-NE suppressed only NS (Wilcoxon test, $z = 4.36$, $p < .00006$). There was no significant change in preening in birds that showed BC suppression alone (Wilcoxon test, $T = 5$, $p > .10$) or no change (Wilcoxon test, $z = 0.47$, $p > .10$).

The nine birds that appeared inactive following 1-NE administration stood immobile in a hunched posture with feathers raised and one or both eyes closed for most of the observation period. All courtship displays were markedly suppressed in these birds (chasing, Wilcoxon test, $T = 0$, $p < .01$). However, as indicated in Table 1, preening was not inhibited (Wilcoxon test, $T = 7$, $p > .10$).

Since BC is usually performed before NS in initial male-female interactions, 1-NE may have acted after a longer latency in birds showing only NS suppression than in those showing both BC and NS suppression. If this were the case, then birds in which NS was inhibited might be

expected to perform fewer bouts of BC during the entire 15-minute observation period and especially after the initial three minutes. This was not observed. The median number of bouts of BC performed during the total observation period in the blank tests was 2 (range 1-5) and in the 1-NE test was also 2 (range 1-6; Wilcoxon test, $n = 21$, $T = 73.5$, $p > .10$). The median number of BC bouts performed after the first three minutes was 1 (range 0-4) in both blank and 1-NE conditions (Wilcoxon test, $n = 15$, $T = 42$, $p > .10$).

Following 1-NE application, two birds ate for the greater part of the observation period, 22 and 18 one-half minute intervals out of 30, respectively. These birds did not court. One ate voraciously for a period of 1 1/2 h and at the end of this time had gained 10 g in weight. This bird was subsequently implanted with phenylephrine and isoproterenol. Phenylephrine elicited only 5 one-half minute intervals of eating whereas isoproterenol produced eating in 20 one-half minute intervals.

Figure 1 portrays the anatomical location of each 1-NE implant. L-NE suppressed courtship predominantly within a ventromedial forebrain region comprising the tuberculum olfactorium (TO), nucleus and fasciculus diagonalis Brocae (FDB), tractus septomesencephalicus (TSM), nucleus preopticus anterior (POA), nucleus preopticus lateralis (POL), stratum cellulare externum (SCE), and nucleus lateralis hypothalami (LHy). L-NE also effectively suppressed courtship in the nucleus septalis lateralis (SL), as did single implants in the commissura

anterior (CA), cerebellum, nucleus rotundus, and fourth ventricle.

Table 2 provides a quantitative analysis of the behaviorally suppressive effects of l-NE by implant localization. It indicates separately the percentage of l-NE implants that inhibited one or more courtship displays (BC or NS), BC (whether or not accompanied by NS suppression), NS (whether or not accompanied by BC suppression), and both BC and NS. The table indicates that the percentage of l-NE implants that reduced the display of courtship was related to the anatomical placement of the drug. Focusing attention first on the nucleus preopticus anterior-nucleus preopticus medialis-nucleus anterior medialis hypothalami (POA-POM-AM) continuum, it can be seen that two of five implants (40%) in this area suppressed courtship. L-NE was most effective in the areas immediately surrounding this POA-POM-AM continuum, i.e., in the FDB-TSM, POL, and SCE-LHy, where 81-100% of implants produced courtship suppression. In the TO, an area further removed anteriorly from the POA-POM-AM continuum, l-NE suppressed courtship in 53% of the birds. Outside the ventromedial forebrain region, l-NE implants in the SL suppressed either BC or NS in 63% of the birds, and single implants in the commissura anterior, cerebellum, nucleus rotundus, and fourth ventricle also suppressed courtship. None of the l-NE implants in the paleostriatum augmentatum (PA), lobus parolfactorius (LPO), lateral ventricle (V), or chiasma opticum (CO) suppressed courtship.

In addition to the relationship between anatomical placement and

Table 2

Percentage of l-Norepinephrine Implants Suppressing Courtship or Activity in Various Brain Regions

Brain Region	No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr	% Inactive
Ventromedial Forebrain	77	75	34	75	34	8
TO[a]	19	53	5	53[a]	5	11
FDB-TSM	31	81	29	81	29	3
POA-POM-AM	5	40	40	40	40	40
POL	10	90	70	90	70	10
SCE-LHy	12	100	58	100	58	0
SL	8	63	38	25	0	0
PA-LPO-V	16	0	0	0	0	0
Others[b]	15	27[c]	27[c]	0	0	20[d]
Total	116	61	28	52	22	8

[a]One implant that suppressed NS also enhanced BC.

[b]N. periventricularis magnocellularis (PVM, n = 3); midbrain reticular formation (n = 3); cerebellum (n = 2); commissura anterior (CA, n = 1); n. rotundus (n = 1); fourth ventricle (n = 1); n. lateralis hypothalami (LHy) at the level of n. medialis hypothalami posterioris (n = 2); chiasma opticum (CO, n = 2).

[c]Single implants in n. rotundus, cerebellum, fourth ventricle, commissura anterior (CA).

[d]All three implants in n. periventricularis magnocellularis (PVM).

degree of effectiveness, Figure 1 and Table 2 also indicate that 1-NE differentially affected BC and NS in relation to its anatomical location. First, implants within the ventromedial forebrain region suppressed NS more than BC, but those outside this region suppressed BC more than NS. Indeed, the only individual implants to suppress BC without also suppressing NS were found outside the ventromedial forebrain. All implants within the ventromedial forebrain that suppressed BC also suppressed NS. The ventromedial forebrain region also contained implants that suppressed only NS, and the two SL implants that suppressed NS alone were very close to this region. Second, the percentage of implants that suppressed NS without also suppressing BC within the ventromedial forebrain increased with the distance of the implant from the POA-POM-AM continuum. Thus, the two implants in the POA that affected courtship suppressed both the BC and NS displays. However, 20% of the implants in the POL and 42-52% of those in the more distant FDB-TSM, SCE-LHy, and TO areas suppressed NS without suppressing BC.

As noted in Table 2, one implant in the TO that suppressed NS also enhanced BC. The implants that produced inactivity were located in the nucleus periventricularis magnocellularis (PVM; n = 3), TO (n = 2), FDB (n = 1), POA (n = 1), AM (n = 1), and POL (n = 1). The two birds that ate considerable quantities of food received drug implants in the nucleus lateralis hypothalami at the level of the nucleus medialis hypothalami posterioris.

d-Norepinephrine

Twenty-one birds that received the d-isomer of norepinephrine showed the following behavioral results: suppression of both BC and NS (n = 2); suppression of NS alone (n = 2); suppression of BC alone (n = 6); enhancement of BC (n = 1); no change (n = 10). As indicated in Table 3, the majority of d-NE implants that suppressed courtship were found in the ventromedial forebrain region. Although d-NE was less effective than l-NE in this region (50% effective d-NE implants vs 75% for l-NE), the ability of d-NE to suppress courtship was apparently also related to its anatomical distance from the POA-POM-AM continuum. However, unlike l-NE, d-NE suppressed BC more than NS in the ventromedial forebrain. It is interesting to note that ventromedial forebrain implants of d-NE suppressed BC in only a slightly larger percentage of birds than did l-NE (38% vs 34%), but the same implants suppressed NS in a much smaller percentage of birds than did l-NE (25% vs 75%).

Phenylephrine

The behavioral results observed upon phenylephrine application in 31 birds were as follows: suppression of BC and NS (n = 5); suppression of NS alone (n = 8); suppression of BC alone (n = 2); enhancement of BC (n = 1); inactivity (n = 1); no change (n = 14). Table 4 indicates that all but one phenylephrine implant that suppressed courtship were found in the ventromedial forebrain region. This alpha-adrenergic receptor agonistic

Table 3

Percentage of d-Norepinephrine Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		16	50	38	25	17
TO	4		0	0	0	0
FDB-TSM	5		40	20	40	20
POA-POM-AM	1		100	100	100	100
POL[a]	6		84	67	17	0
SCE-LHy	0					
SL		1	0	0	0	0
PA-LPO-V		3	33	33	0	0
Others[b]		1	100	100	0	0
Total		21	57	38	19	10

[a]One implant enhanced BC.

[b]Commissura anterior (CA).

Table 4

Percentage of Phenylephrine Implants Suppressing Courtship or Activity in Various Brain Regions

Brain Region	No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr	% Inactive
Ventromedial Forebrain	23	57	30	52	26	4
TO	4	50	25	50	25	25
FDB-TSM	8	63	38	50	25	0
POA-POM-AM	1	0	0	0	0	0
POL	5	60	20	60	20	0
SCE-LHy	5	80	40	60	20	0
SL	2	50	0	50	0	0
PA-LPO-V	4	0	0	0	0	0
Others[a]	2	0	0	0	0	0
Total	31	48	23	42	16	3

[a]One implant in chiasma opticum (CO) enhanced BC; other implant in commissura anterior (CA).

drug, which suppressed courtship in 57% of the birds receiving the drug in the ventromedial forebrain, was also maximally effective in the areas adjacent to the POA-POM-AM continuum. Like l-NE, phenylephrine suppressed NS relatively more than BC in the ventromedial forebrain. Only two implants in this area suppressed BC without also suppressing NS.

Table 4 also indicates that one phenylephrine implant in the TO produced symptoms of inactivity similar to those observed in birds that appeared inactive following l-NE administration.

Isoproterenol

The results of isoproterenol administration in 28 birds were as follows: suppression of BC and NS (n = 3); suppression of NS alone (n = 3); suppression of BC alone (n = 5); enhancement of BC alone (n = 2); and no change (n = 15).

Table 5 indicates that this beta-adrenergic receptor agonist suppressed courtship predominantly within the ventromedial forebrain. However, it was less effective than l-NE in this area, with only 50% of implants suppressing courtship. Furthermore, unlike l-NE and phenylephrine in this area, isoproterenol suppressed BC more than NS and had a relatively large proportion of implants (25%) suppress only BC. The results of ventromedial forebrain placements of isoproterenol are comparable to those of d-NE in the way they differ from the results of l-NE and phenylephrine. Both isoproterenol and d-NE suppressed BC in a

Table 5

Percentage of Isoproterenol Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		20	50	40	25	15
TO	4		50	25	50	25
FDB-TSM	6		67	50	17	0
POA-POM-AM	0					
POL	4		50	50	25	25
SCE-LHy	6		33	33	17	17
SL		2	0	0	0	0
PA-LPO-V[a]		4	25	0	25[a]	0
Others[b]		2	0	0	0	0
Total		28	36	25	21	11

[a]One implant enhanced BC.

[b]One implant in chiasma opticum (CO) enhanced BC; other implant in commissura anterior (CA).

somewhat larger percentage of birds than did l-NE and phenylephrine. Yet they suppressed NS in a much smaller percentage of birds than the two predominantly alpha-adrenergic receptor agonists.

Tyramine

In 13 birds tyramine implants were associated with the following responses: suppression of NS alone (n = 2); suppression of BC alone (n = 3); suppression of BC and enhancement of NS (n = 1); enhancement of BC alone (n = 1); and no change (n = 6). As indicated in Table 6, ventromedial forebrain implants of tyramine, a drug which releases l-NE from nerve endings (Colburn & Kopin, 1972), suppressed courtship in 36% of the birds. As observed with d-NE and isoproterenol, the amount of BC suppression produced by tyramine, 27%, was not very much smaller than that produced by l-NE. However, the amount of NS suppression (9%) was considerably smaller than that produced by l-NE.

The tyramine implant in the FDB that suppressed BC also enhanced NS. One implant in the POL increased BC performance.

Desipramine

The responses observed in 16 birds following desipramine application were: suppression of BC and NS (n = 1); suppression of NS (n = 1); suppression of BC (n = 1); no change (n = 13). Ventromedial placements of this drug, which may increase the concentration of endogenously released NE at receptors by blocking its uptake by nerve endings

Table 6

Percentage of Tyramine Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		11	36	27	9	0
TO	2		50	50	0	0
FDB-TSM[a]	5		40	20[a]	20	0
POA-POM-AM	0					
POL[b]	4		25	25	0	0
SCE-LHy	0					
SL		0				
PA-LPO-V		2	100	50	50	0
Total		13	46	31	15	0

[a]One implant that suppressed BC also enhanced NS.

[b]One implant enhanced BC.

(Glowinski & Axelrod, 1964; Lidbrink, Jonsson, & Fuxe, 1971; Schanberg, Schildkraut, & Kopin, 1967), suppressed courtship in only 14% of the birds (Table 7).

Dopamine

The following results were noted in 14 birds tested with dopamine: suppression of BC (n = 2); enhancement of BC (n = 1); enhancement of NS (n = 1); no change (n = 10). Dopamine had only small variable effects on courtship (Table 8).

Alpha-Methyl-Para-Tyrosine

Fourteen birds received this inhibitor of catecholamine synthesis (Spector, Sjoerdsma, & Udenfriend, 1965). One bird showed BC suppression and another NS suppression (Table 9).

Phentolamine

No changes in behavior were noted in 6 birds that received this alpha-adrenergic receptor antagonist (Table 10).

LB 46

Of eight birds that received this beta-adrenergic receptor antagonist (Giudicelli, Schmitt, & Boissier, 1969; Singh & Vaughan Williams, 1971), seven showed no changes in behavior. One implant in the cerebellum suppressed the BC display (Table 11).

Table 7

Percentage of Desipramine Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		14	14	14	7	7
TO	4		0	0	0	0
FDB-TSM	8		13	13	13	13
POA-POM-AM	0					
POL	2		50	50	0	0
SCE-LHy	0					
SL		1	100	0	100	0
PA-LPO-V		1	0	0	0	0
Total		16	19	13	13	6

Table 8

Percentage of Dopamine Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		11	9	9	0	0
TO	3		0	0	0	0
FDB-TSM[a]	6		33	33	0	0
POA-POM-AM	0					
POL	2		0	0	0	0
SCE-LHy	0					
SL		0				
PA-LPO-V[b]		3	0	0	0	0
Total		14	7	7	0	0

[a]One implant enhanced BC.

[b]One implant enhanced NS.

Table 9

Percentage of Alpha-Methyl-Para-Tyrosine Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		11	18	9	9	0
TO	2		50	50	0	0
FDB-TSM	6		0	0	0	0
POA-POM-AM	0					
POL	2		50	0	50	0
SCE-LHy	1		0	0	0	0
SL		1	0	0	0	0
PA-LPO-V		1	0	0	0	0
Others[a]		1	0	0	0	0
Total		14	14	7	7	0

[a]Chiasma opticum (CO).

Table 10

Percentage of Phentolamine Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		4	0	0	0	0
TO	1		0	0	0	0
FDB-TSM	2		0	0	0	0
POA-POM-AM	0					
POL	1		0	0	0	0
SCE-LHy	0					
SL		0				
PA-LPO-V		1	0	0	0	0
Others[a]		1	0	0	0	0
Total		6	0	0	0	0

[a]Midbrain reticular formation.

Table 11

Percentage of LB 46 Implants Suppressing Courtship
in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		4	0	0	0	0
TO	0					
FDB-TSM	3		0	0	0	0
POA-POM-AM	0					
POL	1		0	0	0	0
SCE-LHy	0					
SL		1	0	0	0	0
PA-LPO-V		1	0	0	0	0
Others[a]		2	50[b]	50[b]	0	0
Total		8	13	13	0	0

[a]Midbrain reticular formation; cerebellum.

[b]Cerebellum.

Serotonin

Serotonin implants were associated with the following behavioral results in 38 birds: suppression of BC and NS (n = 3); suppression of NS alone (n = 5); suppression of BC alone (n = 3); enhancement of BC (n = 1); enhancement of BC and NS (n = 1); inactivity (n = 2); no change (n = 23). Serotonin suppressed courtship predominantly within the ventromedial forebrain and SL (Table 12). Serotonin implants in the ventromedial forebrain were effective in 31% of the birds, and like l-NE, suppressed NS more than BC. The two implants in the SL that produced BC suppression were made in the same birds in which l-NE suppressed BC. Two birds which received serotonin in the TO appeared inactive, displaying the same symptoms as those observed in birds that appeared inactive after receiving l-NE.

Parachlorophenylalanine (PCPA)

PCPA, the synthesis inhibitor of serotonin (Koe & Weissman, 1966), had the following effects in 9 birds: suppression of BC and NS (n = 1); suppression of NS (n = 1); suppression of BC (n = 1); enhancement of BC and NS (n = 1); no change (n = 5). As indicated in Table 13, 29% of the ventromedial forebrain implants of PCPA suppressed courtship, and these had a greater effect on BC than NS.

Barium Chloride

The following behavioral results were noted in 26 birds that

Table 12

Percentage of Serotonin Implants Suppressing Courtship or Activity in Various Brain Regions

Brain Region	No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr	% Inactive
Ventromedial Forebrain	26	31	15	27	12	4
TO	6	17	17	17	17	33
FDB-TSM	10	30	10	30	10	0
POA-POM-AM	1	100	100	0	0	0
POL	5	40	20	40	20	0
SCE-LHy[a]	4	25	0	25	0	0
SL	5	40	40	0	0	0
PA-LPO-V[b]	5	20	0	20	0	0
Others[c]	2	0	0	0	0	0
Total	38	29	16	21	8	5

[a] One implant enhanced BC.

[b] One implant enhanced BC and NS.

[c] Implants in commissura anterior (CA) and chiasma opticum (CO).

Table 13

Percentage of PCPA Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		7	29	29	14	14
TO	0					
FDB-TSM	4		25	25	0	0
POA-POM-AM	1		0	0	0	0
POL	1		100	100	100	100
SCE-LHy[a]	1		0	0	0	0
SL		1	0	0	0	0
PA-LPO-V		1	100	0	100	0
Total		9	33	22	22	11

[a]One implant enhanced BC and NS.

received the vasoconstrictor barium chloride (Rosenblum, 1976): suppression of BC and NS (n = 3); suppression of NS (n = 6); suppression of BC (n = 1); inactivity (n = 8); motor disturbance (n = 1); no change (n = 7). Table 14 indicates that 44% of barium chloride implants in the ventromedial forebrain region and 38% of implants in the PA-LPO-V areas suppressed courtship. NS was suppressed more than BC in both areas.

Barium chloride produced inactivity in a considerably larger proportion of birds than either l-NE, phenylephrine, or serotonin. The inactivity did not differ qualitatively from that induced by the other drugs. The likelihood of observing inactivity seemed to be related to the proximity of the implant to the POA-POM-AM region: 100% of the implants in the POL produced inactivity, and the percentage declined progressively in the more distant areas.

Papaverine

Fifteen birds showed the following responses upon administration of the vasodilator drug papaverine (Lende, 1960): suppression of NS (n = 3); suppression of BC (n = 4); no change (n = 8). As indicated in Table 15, BC or NS were suppressed in 50% of the birds receiving this drug within the ventromedial forebrain. BC suppression was slightly greater than NS suppression.

Gamma-Amino-Butyric Acid (GABA)

Of 12 birds that received this neural inhibitory drug and putative

Table 14

Percentage of Barium Chloride Implants Suppressing Courtship or Activity in Various Brain Regions

Brain Region	No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr	% Inactive
Ventromedial Forebrain	16	44	19	38	13	44
TO 4		50	50	25	25	25
FDB-TSM 7		57	14	57	14	43
POA-POM-AM 0						
POL 2		0	0	0	0	100
SCE-LHy 3		33	0	33	0	33
SL	1	0	0	0	0	0
PA-LPO-V	8	38	13	38	13	13
Others[a]	1	0	0	0	0	0
Total	26	38	15	35	12	31

[a]Implant in cerebellum; bird experienced motor disturbance.

Table 15

Percentage of Papaverine Implants Suppressing Courtship
in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		14	50	29	21	0
TO	4		50	25	25	0
FDB-TSM	9		56	33	22	0
POA-POM-AM	0					
POL	1		0	0	0	0
SCE-LHy	0					
SL		0				
PA-LPO-V		1	0	0	0	0
Total		15	47	27	20	0

neurotransmitter (Curtis & Johnston, 1974; Krnjević, 1970), 11 showed no change in behavior. One showed NS suppression (Table 16).

Glutamic Acid

One out of 10 birds that received this neural excitatory substance and putative neurotransmitter (Curtis & Johnston, 1974; Krnjević, 1970) showed NS suppression. No other behavioral changes were observed (Table 17).

Pre-Surgical vs Post-Surgical Behavior

A general indication of how the behavior of the experimentally prepared male doves (castrated, receiving daily injections of 100 μg of testosterone proprionate, and bearing a chronic brain cannula) related to that of intact doves was obtained by comparing the birds' pre-surgical screening test performance with their post-surgical median blank test performance. The results are given in Table 18. Chasing, BC, and nest-cooing performances were significantly reduced in the post-surgical blank tests compared with the initial screening test (Wilcoxon test: chasing, $z = 3.97$, $p < .0001$; BC, $z = 4.03$, $p < .00006$; nest-cooing, $z = 2.01$, $p < .044$). Wing-flipping was not significantly different post-surgically than pre-surgically (Wilcoxon test, $z = 1.16$, $p > .10$). The level of preening, however, was greater post-surgically than pre-surgically (Wilcoxon test, $z = 2.32$, $p = .02$).

Table 16

Percentage of GABA Implants Suppressing Courtship
in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		7	14	0	14	0
TO	3		0	0	0	0
FDB-TSM	3		0	0	0	0
POA-POM-AM	0					
POL	1		100	0	100	0
SCE-LHy	0					
SL		1	0	0	0	0
PA-LPO-V		4	0	0	0	0
Total		12	8	0	8	0

Table 17

Percentage of Glutamic Acid Implants Suppressing Courtship in Various Brain Regions

Brain Region		No. of Birds	% BC or NS Suppr	% BC Suppr	% NS Suppr	% BC and NS Suppr
Ventromedial Forebrain		8	13	0	13	0
TO	1		0	0	0	0
FDB-TSM	4		25	0	25	0
POA-POM-AM	1		0	0	0	0
POL	2		0	0	0	0
SCE-LHy	0					
SL		0				
PA-LPO-V		2	0	0	0	0
Total		10	10	0	10	0

Table 18

Performance of Courtship Behavior Patterns in Pre-Surgical Screening Test and Post-Surgical Blank Tests (N = 116)

Behavior Pattern	Pre-Surgical Median	Pre-Surgical Range	Post-Surgical Median	Post-Surgical Range	p
	(No. or 1/2-min intervals[a])				
Cha	9.5	1-20	5.5	1-14	< .0001
BC	44	2-192	21	1-103.5	< .00006
WF	9	1-29	9.5	1-26.5	> .10
NC	34	1-198	27	2-115.5	< .044
Pr	3.8	0-26	9.5	0-18	< .02

Note. Cha = chasing; BC = bow-cooing; WF = wing-flipping; NC = nest-cooing; Pr = preening.

[a]BC and NC are expressed in number of times the birds engaged in these behavior patterns. Cha, WF, and Pr are expressed in number of 1/2-min intervals in which the birds engaged in these behavior patterns.

"Unstable" vs "Stable" Implant Locations

The fact that 30 birds omitted either the BC, NS, or both courtship displays on more than 40% of the control blank tests raised the possibility that cannula placement in the brain may have been partly responsible for the behavioral instability observed in these birds. Table 19 indicates the percentage of cannula implants in the various regions of the brain that were associated with "unstable" or "stable" blank performance. In all regions but the POA-POM-AM continuum, 75% or more cannula placements belonged to birds that had met the criterion of behavioral stability. However, of the 19 placements within the POA-POM-AM continuum, 74% belonged to birds that had failed to meet the criterion.

Table 19

Percentage of Birds with Cannulae in Various Brain Regions Showing Stable and Unstable Control Courtship Behavior

Brain Region	No. of Birds	% Stable	% Unstable
TO	21	90	10
FDB-TSM	35	89	11
POA-POM-AM	19	26	74
POL	13	77	23
SCE-LHy	12	100	0
SL	10	80	20
PA-LPO-V	16	100	0
Others	20	75[a]	25[b]

[a] N. periventricularis magnocellularis (PVM, n = 3); midbrain reticular formation (n = 3); cerebellum (n = 2); commissura anterior (CA, n = 1); n. rotundus (n = 1); fourth ventricle (n = 1); n. lateralis hypothalami (LHy) at the level of n. medialis hypothalami posterioris (n = 2); chiasma opticum (CO, n = 2).

[b] N. accumbens (n = 2); n. periventricularis magnocellularis (n = 1); neostriatum caudale (n = 1); n. taeniae (n = 1).

DISCUSSION

Anatomical Results

Intracerebral implants of l-NE most effectively suppressed courtship displays in the male ring dove when they were placed within the SL and a ventromedial forebrain region having the POA-POM-AM continuum as its approximate center. L-NE was maximally effective in the areas adjacent to the POA-POM-AM continuum; 81-100% of implants in the POL, FDB-TSM and SCE-Lhy suppressed courtship. In the more distant TO, 53% of implants suppressed courtship. Within the POA-POM-AM itself, only 40% of the implants suppressed courtship. Outside the ventromedial forebrain, 63% of SL implants inhibited courtship, as did single implants in the commissura anterior, cerebellum, fourth ventricle, and nucleus rotundus. No implants in the PA, LPO, V, or chiasma opticum suppressed the displays.

Implants in the vicinity of the POA-POM-AM also tended to suppress courtship more completely than those further removed. All three courtship displays, chasing, BC, and NS, were suppressed by 29-70% of implants in the POA, POL, FDB-TSM, and SCE-LHy, by 5% of implants in the TO, and by none of the implants outside the ventromedial forebrain.

Partial courtship suppression involved inhibition of only BC or NS.

The apparent anatomical gradient of responsiveness to l-NE was analogous to that found for cerebral testosterone implants in castrated male doves (Hutchison, 1970b, 1971). Hutchison found that testosterone proprionate implants produced maximal restoration of behavior in the vicinity of (and including) the preoptic-anterior hypothalamic region. Implants a short distance from this region restored courtship in a smaller percentage of birds, and reinstated only one display.

In addition to the large suppressive effect of l-NE in the ventromedial forebrain, it was noted that 74% of birds whose cannula implants were located directly in the POA-POM-AM continuum showed courtship suppression in control blank tests. These birds omitted the BC or NS displays on more than 40% of the blank tests. This finding suggests that the lesion created by the presence of the cannula tip in the POA-POM-AM tissue might have suppressed courtship. Since the degree of courtship inhibition by l-NE was generally related to the proximity of the drug implant to this region, l-NE may have exerted its inhibitory effect on courtship by diffusing to and disrupting neural activity within the POA-POM-AM. It has been shown that crystalline l-NE applied to the hypothalamus of the rat in the same manner as administered in the present study diffuses from the tip of the cannula in a sphere having a diameter of 1.0-1.8 mm (Grossman & Stumpf, 1969; Kent, 1972).

A further relationship was observed between the anatomical

placement of l-NE and its differential effectiveness on BC and NS. The NS display was suppressed to a greater degree than BC in all areas of the ventromedial forebrain with the exception of the POA (where both displays were suppressed equally). Moreover, the ratio of NS to BC suppression increased with the distance of the l-NE implant from the POA-POM-AM continuum. On the other hand, l-NE implants in the SL suppressed BC more than NS, and those in the commissura anterior, cerebellum, fourth ventricle, and nucleus rotundus suppressed only BC.

The fact that several l-NE implants in the SL and adjacent commissura anterior suppressed BC without also suppressing NS, a response never observed in the ventromedial forebrain (where BC suppression was always accompanied by NS suppression), suggests that the SL may contain a noradrenergic neural system that inhibits BC selectively. A lesion study by Cooper and Erickson (1976) has provided evidence for the existence of a NS-inhibitory neural system in the SL that may be located posterior to the BC system implicated in the above observations.

The larger degree of NS than BC suppression by l-NE implants in the ventromedial forebrain has two possible explanations: (a) Since BC is usually performed before NS in initial male-female interactions, l-NE from the peripheral ventromedial forebrain implants would reach the BC and NS neural system contained in the POA-POM-AM region in a concentration sufficient to suppress the displays only after a large amount of BC had already been displayed. Thus, a greater suppressive effect on

NS would be observed. This explanation would predict that 1-NE implants which suppressed NS alone would also produce fewer bouts of BC during the total observation period and especially after the first three minutes, the time when 1-NE's effectiveness was generally being observed on NS. This prediction, however, was not confirmed. (b) NS may have a lower threshold for suppression by 1-NE than does BC, a situation analogous to NS's apparently lower threshold for reinstatement by testosterone in castrated birds (Hutchison, 1970b). This hypothesis is suggested by the similarity between the neuroanatomical patterns of differential effectiveness on BC and NS produced by 1-NE and testosterone. As with 1-NE, Hutchison (1970b, 1971) found that testosterone implants in the ventromedial forebrain affected (i.e., restored) NS more readily than BC and that more peripheral implants acted only upon NS. Thus, the greater degree of NS suppression induced by 1-NE implants in the ventromedial forebrain may have been due to the fact that the lower concentration of 1-NE arriving at the POA-POM-AM from many of the peripheral implants exceeded the threshold for NS suppression but not BC suppression. However, this hypothesis may apply only to a neural system that is specifically activated by 1-NE and phenylephrine since most of the other drugs that produced large amounts of courtship suppression did not suppress NS to a greater degree than BC in the ventromedial forebrain. Two other drugs that did produce more NS than BC suppression in the ventromedial forebrain, serotonin and barium chloride, had anatomical patterns of

differential responsiveness on BC and NS that were not consistent with the above hypothesis.

One l-NE implant, as well as one or two implants of the other sympathomimetic drugs, serotonin and PCPA, enhanced the performance of courtship displays. However, these implants lacked any systematic neuroanatomical distribution.

Several l-NE, phenylephrine, serotonin, and barium chloride implants produced a generalized depressant effect in the birds characterized by immobility, hunched posture, raised feathers, and closed eyes. While only a small percentage of l-NE (8%), phenylephrine (3%), and serotonin (5%) implants produced this behavioral inactivity, 31% of barium chloride implants produced inactivity. Although the majority of implants that produced inactivity were located in areas that also produced courtship suppression, three l-NE implants in the nucleus periventricularis magnocellularis induced inactivity.

Marked eating was elicited by l-NE and isoproterenol implants in the nucleus lateralis hypothalami at the level of the nucleus medialis hypothalami posterioris. This response may be specifically dependent upon beta-adrenergic receptor stimulation in this area since phenylephrine application did not induce any unusual amount of eating.

Physiological Nature of Courtship Suppression

A large number of pharmacologically heterogeneous drugs suppressed courtship displays in the ring dove. These drugs, in descending

order of the percentage of their implants in the ventromedial forebrain that suppressed BC or NS, include: l-NE (75%) > phenylephrine (57%) > d-NE (50%) = isoproterenol (50%) = papaverine (50%) > barium chloride (44%) > tyramine (36%) > serotonin (31%) > PCPA (29%) > alpha-methyl-para-tyrosine (18%) > desipramine (14%) = GABA (14%) > glutamic acid (13%) > dopamine (9%). No phentolamine or LB 46 implants in the ventromedial forebrain suppressed courtship. These results suggest that courtship was most readily suppressed by increases in noradrenergic activity produced by the directly acting sympathomimetic drugs and presumably also by the changes in vasomotor function produced by papaverine and barium chloride. However, the increased noradrenergic activity was neither stereospecific for NE nor specific for alpha or beta adrenergic receptor stimulation. Similarly, the change in vasomotor function was apparently not specific for either vascular contraction or dilatation.

Courtship was moderately suppressed by the indirectly acting sympathomimetic tyramine and by either increased or decreased levels of serotonin. Although the increased serotonin levels achieved by application of serotonin and the decreased serotonin levels achieved by application of PCPA would not be expected to have the same effect on behavior, there is other evidence that serotonin and PCPA have the same physiological effect in several mammalian tissues (Marley & Whelan, 1974).

Very little or no courtship suppression occurred following inhibition of catecholamine synthesis by alpha-methyl-para-tyrosine, blockade of

the uptake of NE by desipramine, generalized neural inhibition by GABA, generalized neural excitation by glutamic acid, increased dopamine levels by its direct application, blockade of alpha-adrenergic receptors by phentolamine, and blockade of beta-adrenergic receptors by LB 46. The small suppressive effect produced by desipramine may indicate either that endogenous NE does not inhibit courtship, or if it does so, that it is not released in any significant quantities when a bird is in a situation that elicits courtship behavior. It is also possible that the alpha-adrenergic receptor blocking action of desipramine (Brodie, Dick, Kielholz, Pöldinger, & Theobald, 1961; Hughes, Kneen, & Main, 1974) may have been of sufficiently large magnitude in the ring dove to offset the effect of increased NE levels at the receptor sites.

Although the large variability in drug effectiveness could be evidence for a neurochemically specific system that regulates courtship behavior, the large degree of pharmacological nonspecificity and the anatomical findings indicating that lesions of the POA-POM-AM inhibit courtship performance suggest that nonspecific effects of the drugs may have been largely responsible for the observed courtship suppression. The degree of effectiveness of any particular drug may have been related simply to its ability to disrupt normal functional activity in the POA-POM-AM region of the brain.

The behaviorally suppressive effects of barium chloride and papaverine suggest that the nonspecific effects of the monoamines may have

included vascular changes. Many studies have shown that topical application of NE, serotonin, tyramine, isoproterenol and phenylephrine may either constrict or dilate cerebral arteries depending on the dose and vessel (Allen & Gross, 1976; Allen, Henderson, Chou, & French, 1974; Deshmukh & Harper, 1971; Edvinsson & Owman, 1974; Lende, 1960; MacKenzie, McCullough, & Harper, 1976; Mitchell, Scriven, & Rosendorff, 1975; Raynor, McMurtry, & Pool, 1961; Rosenblum, 1976; Rosendorff & Cranston, 1971; Toda, 1976; Wahl, Kuschinsky, Bosse, & Neiss, 1974). However, whether the behavioral results of papaverine indicate that vascular dilatation suppresses courtship cannot be ascertained since papaverine also inhibits the enzyme phosphodiesterase (Bloom, Siggins, Hoffer, Segal, & Oliver, 1975; Kukovetz & Pöch, 1970). Although papaverine does not appear to have this property in the chicken brain (Nahorski & Rogers, 1976), such a pharmacological effect in the ring dove would increase levels of cyclic AMP, a substance that may be the intracellular second messenger of noradrenergic, serotonergic, and dopaminergic effects induced in neurons (Bloom, Siggins, Hoffer, Segal, & Oliver, 1975; Dismukes & Mulder, 1976; Rall & Sattin, 1970; Satoh, Satoh, Notsu, & Honda, 1976).

Another possible way in which the drugs may have produced courtship suppression is by altering other physiological functions regulated by the POA-POM-AM region of the brain whose imbalance may have indirectly affected courtship. Six of the nine drugs having the largest

suppressive effects on courtship, l-NE, d-NE, phenylephrine, isoproterenol, tyramine, and serotonin, have been found to produce behavioral and EEG sleep and changes in body temperature, blood pressure, and oxygen consumption when injected into the hypothalamus in the adult chicken or intravenously in young chickens in which the blood-brain-barrier is not developed (Dewhurst & Marley, 1965; Marley & Nistico, 1972, 1975; Marley & Stephenson, 1970).

The individual instances of inactivity observed with some barium chloride, l-NE, phenylephrine, and serotonin implants may have represented merely the more severe cases of generalized behavioral depression produced in varying degrees by all drug implants. The milder cases may have been detectable only in chasing, BC, and NS behavior. Preening, however, was not depressed in any birds receiving l-NE, including those that appeared inactive. In fact, preening increased significantly in birds showing either NS suppression or suppression of both BC and NS. (An inverse relationship between the level of BC and NS performance, on the one hand, and that of preening, on the other, has been observed in behavioral comparisons of intact and castrated birds [Erickson, 1965] and of intact males displaying low and high levels of courtship displays [Cascione, unpublished observations].) The fact that none of the anti-adrenergic drugs, alpha-methyl-para-tyrosine, phentolamine or LB 46, significantly enhanced any behavioral component of courtship provides further support for the notion that the courtship suppression produced by

at least the sympathomimetic amines was due largely to nonspecific effects.

However, if nonspecific effects of the drugs were acting exclusively on BC and NS, then each drug would be expected to either suppress BC and NS equally, or in the case that the two displays had different thresholds for suppression, each drug would have produced approximately the same ratio of NS to BC suppression. Any large difference in the ratios among the drugs might imply that some pharmacologically more specific factors were acting on one display but not the other. The ratios of NS to BC suppression generated by the nine drugs having the largest courtship suppressive effects varied from a high of 2.34 to a low of 0.48. An examination of the data to determine the source of the large variability in ratios reveals that it was due largely to the rather broad range of NS suppression produced by the nine drugs. Whereas the range of BC suppression produced by these drugs was only 15-40%, that of NS suppression was 9-75%. The fact that these drugs had fairly uniform effects on BC but quite variable effects on NS suggests that NS may be selectively regulated by a pharmacologically specific neural system.

It is interesting to speculate that if the highest amount of BC suppression produced by any of the drugs, i.e., 40%, is used as a measure of the maximal amount of courtship suppression that can be produced by the nonspecific action of these drugs on the POA-POM-AM continuum, then only two drugs suppressed NS in excess of this amount. L-NE and

phenylephrine, both predominantly specific alpha-adrenergic receptor agonists, suppressed NS in 75% and 52%, respectively. Thus, in addition to producing nonspecific suppressive effects on both BC and NS, l-NE and phenylephrine may have activated an alpha-adrenergic neural system in the POA-POM-AM that may be selectively involved in the suppression of NS. There is evidence that in avian species NE is found in the hypothalamus in higher concentrations than in any other brain region (Juorio & Vogt, 1967), and the histochemical fluorescence technique has revealed abundant catecholaminergic fibers in the preoptic area (Fuxe & Ljunggren, 1965).

As suggested by the anatomical pattern of responsiveness to l-NE and phenylephrine, the specific alpha-adrenergic component of NS suppression may have a lower threshold than the BC and NS suppression induced by the nonspecific effects of l-NE and phenylephrine. Thus, the lower concentration of l-NE to reach the POA-POM-AM region from the peripheral ventromedial forebrain implants was more likely to suppress NS (activation of the specific alpha-adrenergic component) but the higher concentration to reach this region from closer implants produced nonspecific effects on both BC and NS that would mask the pharmacologically more specific effect.

The present study suggests that the hypothetical alpha-adrenergic neural system involved in the regulation of NS may exert an asymmetrical control over this courtship display. Although stimulation of alpha-

adrenergic receptors by l-NE and phenylephrine inhibited the display, blockade of the receptors by phentolamine did not increase the level of performance of the display. Evidence that some neurotransmitters may have an asymmetrical effect on other neurological functions has been presented by Matthysse (1975). In the ring dove, it is possible that low alpha-adrenergic receptor activity normally predominates under conditions in which the male is ready to court. A further decrease in this activity would be unable to facilitate the performance of NS. However, an increase in alpha-adrenergic receptor activity would reduce behavioral performance. The question of whether a decrease in alpha-adrenergic receptor activity could increase NS performance under conditions in which the male would not be expected to perform at optimal levels could not be answered in the present study. Although the males' performance of nest-cooing in the control blank tests was significantly less than in the pre-surgical screening test, their wing-flipping performance was not significantly different. Thus, the conditions of the present experiment could only reveal a drug's ability to increase NS performance above normal levels, but not its ability to raise NS performance from lower to normal levels.

Tyramine had a much smaller suppressive effect on NS than BC, a finding not consistent with its pharmacological effect of releasing NE from synaptic endings (Colburn & Kopin, 1972). However, this finding may have been due to a combination of tyramine's actions: its ability to

release NE as well as dopamine (VonVoigtlander & Moore, 1973), both of which are found in the ventromedial forebrain region of the avian brain (Juorio & Vogt, 1967), its possible metabolism to dopamine (Muscholl, 1966), and its possible direct receptor stimulating effect (Stoof, Liem, & Mulder, 1976).

The evidence that NS may be selectively suppressed by alpha-adrenergic receptor stimulation might provide a new means of examining the functional significance of the individual courtship displays of the male dove. Several studies indicate that the male's courtship has a profound effect on female physiology and behavior (Erickson, 1970; Erickson & Lehrman, 1964; Hutchison & Lovari, 1976; Lambe & Erickson, 1973). However, in these studies it was impossible to examine the effect of the individual courtship displays on the female response. The technology developed in the present study may be employed to generate males that will not display NS behavior. By evaluating the response of females exposed to these males, a better understanding may be obtained of how the individual components of male courtship behavior help to integrate male and female participation in the reproductive cycle.

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